

# USANA Technical Bulletin

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## Hypercholesterolemia

### Description

- Cholesterol is a necessary component of metabolism. It is widely distributed in the nerve tissues of the brain and spinal cord, and also in the liver, kidneys and adrenal glands. Cholesterol can be synthesized in the liver and is a normal constituent of bile.<sup>1</sup>
- Hypercholesterolemia is characterized by high levels of lipids (especially cholesterol) in the blood.
- Cholesterol circulates in the blood attached to transport molecules known as lipoproteins. These lipoproteins are composed of fat and cholesterol molecules linked to protein. The higher the proportion of cholesterol, the lower the density of the lipoprotein. There are six major classes of lipoprotein particles, the three most commonly identified lipoproteins are: very low density (VLDL), low density (LDL) and high density (HDL).
- LDL transports cholesterol to the cells, and HDL transports cholesterol from the cells for metabolism and excretion. The ratio of HDL to LDL determines whether cholesterol is being deposited in tissues, or broken down and excreted. An elevated LDL level is considered a risk factor for developing cardiovascular disease, stroke and high blood pressure.<sup>2</sup>

### Causes

- Genetic factors can predispose some individuals to hypercholesterolemia.<sup>3</sup>
- Secondary causes of hypercholesterolemia include obesity, diet, lifestyle, excess alcohol, metabolic disorders, other diseases, and medications.<sup>4</sup>

### Prevention and Management

- Dietary modification is recommended as the first step in the treatment of hypercholesterolemia. Current data suggest that dietary fat should be limited to 30% of the total caloric intake, saturated fat should be less than 10% and cholesterol less than 300mg a day.<sup>5</sup>
- Numerous studies show the benefits of fiber in reducing serum cholesterol.<sup>6</sup> The American Heart Association recommends 25 to 30 grams of fiber per day along with the above recommended intakes for fat and cholesterol.<sup>7</sup>
- Antioxidant vitamins such as beta-carotene, Vitamin C, and Vitamin E help to reduce the susceptibility of low density lipoprotein (LDL) to oxidation.<sup>8</sup>

- Therapeutic doses of niacin have been shown to be effective in increasing high-density lipoprotein cholesterol concentrations and reducing the Lp(a) lipoprotein level.<sup>9</sup>
- Impaired vasodilation in humans with hypercholesterolemia has been shown to improve with Vitamin C.<sup>10</sup>

## Abstracts

***Crouse JR 3<sup>rd</sup>. New developments in the use of niacin for treatment of hyperlipidemia: new considerations in the use of an old drug. Coron Artery Dis 1996 Apr; 7(4):321-6.***

Niacin has been used for many years to treat hyperlipidemia. It has been shown to reduce coronary death and non-fatal myocardial infarction and, in a separate analysis of long-term (15-year) follow-up, all cause mortality. It reduces total cholesterol, low density lipoprotein cholesterol (LDL-C) and triglycerides and increases high density lipoprotein cholesterol (HDL-C). Sustained-release niacin may be associated with more dramatic changes in LDL-C and triglyceride, whereas the short acting preparation causes greater increases in HDL-C. The increase of HDL-C occurs at a lower dose (1500 mg/day) than the reduction of LDL-C (> 1500 mg/day). Niacin also favorably influences other lipid parameters including lipoprotein(a) [Lp(a)], alimentary lipemia, familial defective apolipoprotein B-100 and small dense LDL. Combination of niacin with a bile acid sequestrant or a reductase inhibitor represents a powerful lipid-altering regimen. Whereas the reductase inhibitors and bile acid binding resins primarily affect LDL-C, the combined therapy has a synergistic effect to reduce LDL-C and, in addition, the niacin reduces triglycerides and increases HDL-C. The major drawback in the use of niacin is associated side effects (flushing and palpitations) and toxicity (worsening of diabetes control, exacerbation of peptic ulcer disease, gout, hepatitis). Niacin has a long history of use as a lipid lowering agent and has several attractive features. Unfortunately, the side effect profile of this agent warrants its use only in patients with marked dyslipidemia in whom side effects and potential toxicity are closely monitored.

***Rimm EB, Stampfer MJ. The role of antioxidants in preventive cardiology. Curr Opin Cardiol 1997 Mar; 12(2):188-94.*** Vitamin C, carotenoids, and vitamin E, the three main dietary sources of antioxidants, each effect lipid peroxidation and may reduce atherogenesis and lower the risk of coronary heart disease (CHD). Crosscultural studies of antioxidants find that regions with relatively low dietary intake tend to have higher rates of CHD, but in these studies it is difficult to account for other important cardiovascular risk factors. Evidence from observational studies with more detailed information do not support a cardiovascular benefit for vitamin C, although the cardiovascular effect of vitamin C supplementation among populations with marginal vitamin C deficiency is not known. Results from recent clinical trials of beta-carotene supplementation show no cardiovascular benefit, although several observational studies have found an inverse association between carotenoid intake or plasma levels and risk of CHD. The benefit reported in the observational studies may be due to consumption in foods rich in beta-carotene rather than the beta-carotene itself. The evidence for a cardiovascular benefit of antioxidants is strongest for vitamin E. Three large prospective studies find that vitamin E supplement users have approximately 40% lower rates of CHD. Short durations and doses of less than 100 IU/d (when data were available) have no significant effect. The effect of dietary vitamin E may be more modest but still associated with lower risk of CHD in populations in which vitamin E supplementation is infrequent. In a large randomized trial, a nonsignificant reduction in CHD risk was reported for 50 IU/d, although the dose may have been insufficient. A secondary prevention trial of 400 and 800 IU/day reported a strong reduction in nonfatal myocardial infarction, further supporting the large body of evidence that suggests that high doses of vitamin E reduce risk of CHD.

## References

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- <sup>7</sup> Van Horn L. Fiber, lipids, and coronary heart disease: A statement for healthcare professionals from the nutrition committee, American Heart Association. Circulation 1997;95:2701-2704.
- <sup>8</sup> Gilligan DM, Sack MN, Guetta V, Casino PR, Quyyumi AA, Rader DJ, Panza JA, Cannon RO 3<sup>rd</sup>. Effect of antioxidant vitamins on low density lipoprotein oxidation and impaired endothelium-dependent vasodilation in patients with hypercholesterolemia. J Am Coll Cardiol 1994 Dec; 24(7):1611-7.
- <sup>9</sup> Crouse JR 3<sup>rd</sup>. New developments in the use of niacin for treatment of hyperlipidemia: new considerations in the use of an old drug. Coron Artery Dis 1996 Apr;7(4):321-6.
- <sup>10</sup> Ting HH, Timimi FK, Haley EA, Roddy MA, Ganz P, Creager MA. Vitamin C improves endothelium-dependent vasodilation in forearm resistance vessels of humans with hypercholesterolemia. Circulation 1997 Jun 17; 95(12):2617-22.